present specification. The specification has been amended to correct an obvious typographical error.

On page 1 of the Office Action, the Examiner indicates that Claims 1-3, 5-8, 16 and 19-21 are pending; Claims 1, 3, 16 and 19-21 are allowed, and Claims 2 and 5-8 are rejected.

However, the Examiner has failed to set forth the status of Claims 17 and 22, which Applicants also believe should be allowed, since they are directly or indirectly dependent on allowed Claim 16.

On page 2 of the Office Action, the Examiner states that the inventors' original declaration is defective because it does not claim priority to PCT/JP/01512. The Examiner requests the submission of a new oath or declaration which references PCT/JP/01512.

Accordingly, Applicants submit herewith a Substitute Declaration and Power of Attorney which claims priority to PCT/JP99/01512.

At page 3 of the Office Action, the Examiner rejects Claims 5-8 under 35 U.S.C. § 112, first paragraph. Specifically, the Examiner contends that the specification does not sufficiently describe a DNA molecule which hybridizes under stringent conditions to the DNA molecule of SEQ ID NO:2.

Applicants hereby cancel Claims 5 and 8, and amend Claims 6 and 7 to set forth the minimum size of the DNA molecule, thereby rendering moot the Examiner's rejection.

On page 4 of the Office Action, the Examiner rejects Claims 2 and 5-8 under 35 U.S.C. § 102(b) over Fu et al (*PNAS* 1995). Specifically, the Examiner states that Fu et al teaches

that just about any conceivable DNA primer or probe can hybridize, under stringent conditions, to SEQ ID NO:2.

For the following reasons, Applicants respectfully traverse the Examiner's rejection.

Initially, the Examiner is requested to note that Claims 5 and 8 have been cancelled.

Fu et al describes the use of specially designed DNA molecules (partially duplex probes containing single-stranded 3' overhangs) for use in sequencing by hybridization. This reference describes "stacking interactions" which allows a minimum of 5-bases of terminal sequence for priming for Sanger sequencing (see Abstract).

Fu et al does not teach or suggest Applicants' claimed stringent hybridization conditions. The type of hybridization described in Fu et al, i.e., annealing for sequencing reactions, is far removed from the claimed stringent hybridization. Furthermore, there is nothing in Fu et al which teaches a DNA molecule comprising 10-35 nucleotides hybridizing to SEQ ID NO:2.

In regard to Claim 2, the Examiner states that a DNA primer could bind to the transcriptional start site, and thus inhibit or control transcription. Once again, the Examiner's reliance on Fu et al is believe to be misdirected. The Examiner appears to base her theory, at least partially, on personal knowledge and speculation which can not legally be given any weight. Regardless of the weight to be carried by the Examiner's speculative statements, Claim 2 recites a DNA molecule which codes for a protein having transcriptional control activity. There is nothing in Fu et al which describes a DNA molecule

coding for any protein, let alone a protein with transcriptional control activity.

Accordingly, Applicants respectfully submit that the present invention is not taught or suggested by Fu et al, and thus request withdrawal of the Examiner's rejection.

In view of the amendments to the specification, claims and arguments set forth above, reexamination, reconsideration and allowance are respectfully requested.

The Examiner in invited to contact the undersigned at the below-listed number on any questions which might arise.

Respectfully submitted,

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APPENDIX

VERSION WITH MARKINGS TO SHOW CHANGES MADE

IN THE SPECIFICATION:

The specification is amended as follows:

Page 1, line 10 to Page 2, line 2, are changed as follows:

"The p53 protein was discovered as a nuclear protein binding to the large T antigen of the DNA tumor virus SV40 and its gene (p53 gene) has been cloned. At first, the p53 gene was considered to be an oncogene because the transfer of this gene and the ras gene together into cells resulted in transformation of embryonal cells. Later studies, however, revealed that the initially cloned p53 gene was a mutant type and that the wild type rather suppressed the transforming activity of the [multant] mutant type. By now, deletions or anomalies in the p53 gene have been detected in many human cancers and a gamate mutation of the p53 gene was also discovered in Li-Fraumeni syndrome which is known to be a hereditary disease with a high risk for malignant conversion. Because of these and other findings, the p53 gene has by now been considered to be an important suppressor oncogene (Baker, S. J., et al., Science, 244, 217-221 (1989): Nigro, J. M., Nature, 342, 705-708 (1989))."

IN THE CLAIMS:

Claims 5 and 8 are being cancelled.

Claims 6-7 are amended as follows:

Claim 6. (Twice Amended) An isolated DNA molecule comprising 10 to 35 nucleotides which hybridizes under stringent conditions with a DNA molecule comprising nucleotides 145-1488 of SEQ ID NO:2.

Claim 7. (Twice Amended) [A] An isolated DNA [primer] molecule comprising [the isolated DNA molecule of Claim 5] 10 to 35 nucleotides which hybridizes under stringent conditions with a DNA molecule comprising nucleotides 1-2186 of SEQ ID NO:2.